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Identification of a novel signal transduction surface molecule on human cytotoxic lymphocytes.

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Valiante NM, Trinchieri G.

56 Nicholas M Giorgio

Wistar Institute of Anatomy and Biology, Philadelphia, Pennsylvania 19104.

In this study, we have used a newly generated monoclonal antibody (mAb C1.7) to identify a novel 38-kD signal-transducing surface molecule (p38) expressed by lymphocyte subsets capable of cell-mediated cytotoxicity. Virtually all CD16+/CD56+ natural killer (NK) cells and approximately half of CD8+ (T cell receptor [TCR] alpha/beta+) T cells and TCR-gamma/delta+ T cells express the p38 surface molecule. Stimulation of p38 on NK cells with mAb C1.7 activated cytotoxicity, induced lymphokine production, and initiated polyphosphoinositol turnover and [Ca2+]i increases. Unlike other NK cell surface molecules that activate cytotoxicity, p38 stimulation did not result in the release of the granule enzyme N-carbobenzoxy-L-thiobenzyl ester-esterase even under conditions in which mAb C1.7 induced NK cell-mediated redirected lysis of Fc gamma R+ target cells. Activated (recombinant interleukin 2 [rIL-2], 5 d) CD8+ T cells mediated non-major histocompatibility complex (MHC)-restricted cytotoxicity, and the CD8+/p38+ subset contained the overwhelming majority of this activity. F(ab')2 fragments of mAb C1.7 inhibited non-MHC-restricted cytotoxicity mediated by resting NK cells and rIL-2-cultured T cells but did not affect spontaneous cytotoxicity mediated by activated, cultured NK cells. Taken as a whole, our results suggest that p38 may have a direct role in the recognition, signal transduction, and/or lytic mechanisms of non-MHC-restricted cytotoxicity.

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